Increased loop gain mediates the association between smoking and OSA severity

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Objective: Smoking is a known risk factor for obstructive sleep apnea (OSA). However, the specific pathological mechanism linking smoking to OSA remains unclear. This study aims to explore the endotypic traits of OSA among current, former, and non-smokers.

Methods: We prospectively collected polysomnographic data from 980 patients with an apnea-hypopnea index (AHI) ≥ 15 h-1 from a single clinical sleep center. Smoking status was determined through self-reported questionnaires completed prior to polysomnography. Endotypic traits—namely arousal threshold, collapsibility, loop gain, and upper airway compensation—were estimated using polysomnographic signals. Adjusted multivariate linear regression analysis was conducted to investigate the association between smoking and endotypic traits. Causal mediating models were employed to assess the mediating role of endotypic traits between smoking and AHI.

Results: Compared to non-smokers, current smokers exhibited a 7.6 h-1 higher AHI, an 8.8 %eupnea higher arousal threshold, a 4.0 %eupnea worse collapsibility, and a 0.03 higher loop gain during non-rapid eye movement (NREM) sleep. During REM sleep, current smokers showed an 11.1 %eupnea higher arousal threshold and a 0.05 higher loop gain, while former smokers exhibited a 5.7 %eupnea lower upper airway compensation compared to non-smokers. Loop gain modestly mediated the impact of current smoking on AHI (29% in NREM and 40% in REM sleep). Smoking amount and duration since quitting were not linearly associated with AHI or endotypic traits.

Conclusions: Smoking is associated with increased severity of OSA, potentially mediated by elevated loop gain. These effects may be reversible following smoking cessation.

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